Paradoxical septal motion: A diagnostic approach and clinical relevance

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Abstract

Abnormal septal motion (commonly referred to as septal bounce) is a common echocardiographic finding that occurs with several conditions, including the following: mitral stenosis, left bundle branch block, pericardial syndromes and severe pulmonary hypertension. We explore the subtle changes that occur on M-mode imaging of the septum, other associated echocardiographic features, the impact of inspiratory effort on septal motion and relevant clinical findings. Finally, we discuss the impact of abnormal septal motion on cardiac form and function, proposing there is a clinically significant impact on biventricular filling and ejection.

Keywords: cardiac pacing, cardiac surgery, cardiology.

Introduction

The interventricular septum has a characteristic pattern of motion throughout the cardiac cycle (see Figure 1 and Video S1): with thickening and motion away from the transducer (posterior movement or towards the left ventricle) after the onset of electrical depolarisation (QRS complex) followed by a brief 'shudder' at end systole or early diastole coinciding with the end of ventricular repolarization (T wave) and earlier opening of the tricuspid valve and right ventricle filling compared to the left. Finally, the septum returns to its original thickness and position moving back towards the transducer (anterior movement or towards the right ventricle) during diastole.

A multitude of disorders can create an abnormal, paradoxical septal motion, often referred to as 'septal bounce', where the interventricular septum movement is atypical for the particular phase of the cardiac cycle. Although non-diagnostic in itself, interrogation of the specific septal movement can help recognise these conditions and heralds the need for further interrogation. Once identified, a systematic approach is important to delineate the precise cause including the effect of respiratory dynamics. Identification of the precise timing in the cardiac cycle when the septal bounce occurs is important and this may be difficult to delineate with standard 2D imaging and the use of M-mode may be helpful.

Several recent theories have highlighted the important role of the septum in myocardial performance in terms of systolic and diastolic function, in both ventricles, underlying the clinical relevance of abnormal septal motion. This review article aims to describe the causes of septal bounce, a diagnostic approach if it is present and finally describe the role that the septum is proposed to play in cardiac mechanics.

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Causes of abnormal septal motion or septal bounce

Abnormal septal motion can be seen in a variety of clinical conditions, both of cardiac and respiratory aetiology (see Table 1). The more common causes include conduction delays and cardiac surgery patients. In other conditions, paradoxical septal movement may only appear at the extremes, that is: increase in right ventricle (RV) afterload or mitral stenosis.

Diagnostic approach to septal bounce

Various specific patterns of septal motion are described in the literature; however, it is often difficult to discern, necessitating the need for a systematic approach, taking into account respiratory variation, interventricular conduction delay and timing with cardiac cycle, in addition to the clinical setting and related echocardiographic features (see Figure 2).

A useful initial division is based on whether a relationship exists between septal bounce and the respiratory cycle or whether the septal bounce remains constant throughout the respiratory cycle. Respiratory variation in the septum highlights the changing in loading conditions with inspiration and expiration (see Video S2) that are exaggerated in certain conditions. The next step is to identify the timing and duration of the abnormal septal motion within the cardiac cycle (see Table 1). In addition, the ECG should be interrogated for the presence of a cardiac conduction defect.

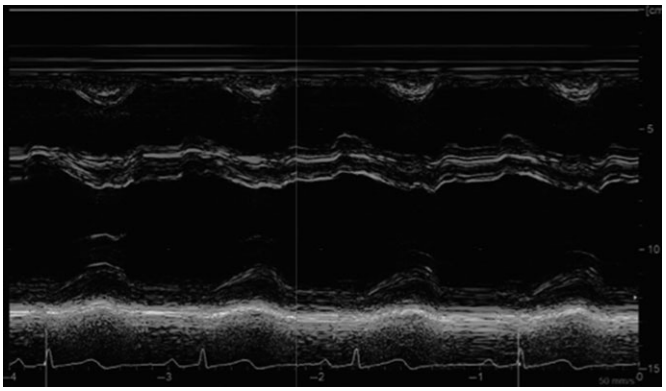

Figure 1: Normal Septal Motion Interrogated with M-mode Imaging.

M-mode echocardiography, especially in the parasternal views, is particularly useful for analysing the septum. By providing high temporal resolution of each part of the septum, it allows analysis of its relationship between the left ventricle lateral wall and the right ventricle throughout the cardiac cycle. Translational error should be avoided, particularly as the direction of motion of the septum may differ in the apex relative to the base.[1] The anterior motion of the right ventricular wall may be exaggerated, and the posterior motion of the septum underplayed due to the relative movement of the heart anteriorly.[1]

Specific causes of abnormal septal motion or septal bounce

Left bundle branch block

With left bundle branch block (LBBB), the normal depolarisation of the septum from left to right is reversed resulting in the RV being depolarised by the right bundle initially, then the LV via the septal branches. This results in dyssynchrony, creating high-amplitude oscillations of the septum.[2] These oscillations result from discordant contraction and ventricular filling. RV contraction occurs earlier than the left, and the septum is displaced posteriorly in early systole (described as 'septal beaking'[3] due to the increase in RV pressure relative to the left (reversal of transeptal pressure gradient).[4] Walmsley *et al.* referred to this same movement to as 'septal flash', and noticed it larger the longer the delay between the right ventricle free wall and septal conduction although felt it occurred irrespective of the pressure gradient between the right and left ventricles.[5] Subsequently, the septum flattens (paradoxical motion) when both ventricles are contracting, before being displaced anteriorly as the left ventricle continues to contract after pulmonic closure. Diastole sees the tricuspid valve open before the mitral valve, and hence the septum is displaced posteriorly, before being reversed, and further displaced anteriorly towards the right ventricle during atrial systole (see Figure 3 and Video S3).

Table 1: Causes of septal bounce.

Cardiac conditions	1 Left bundle branch block 2 'Open' cardiac surgery 3 Septal ischaemia 4 Mitral stenosis 5 Pericardial disease: • Constrictive pericarditis • Tamponade 6 Others: • Congenital absence of pericardium
Pulmonary-related conditions	7 Increase right ventricle afterload • Pulmonary embolism • Increased intrathoracic pressure 8 Increase RV afterload and preload • Asthma/COPD
Others	9. Right ventricle volume overload

Cardiac surgery with full pericardotomies ('open' thoracotomy)

Septal paradoxical motion is a relatively common outcome after open cardiac procedures with anterior motion during systole (see Figure 4 and Video S4). Its occurrence ranges between 30% and 100% shortly after on-pump coronary artery bypass grafting.[6] In a study of 3300 adult patients post-cardiac surgery, 40% developed septal bounce, with no mention of any other form of septal dysfunction (i.e. akinesis, hypokinesis). Other studies have suggested that septal bounce is more likely after valvular repairs or on-pump surgery.[7] The exact aetiology and significance of septal bounce post-cardiac surgery are uncertain, and the postulated mechanisms including septal ischaemia, cold cardioplegia, transient RV dysfunction and may vary with the extent of pericardial incision. Alternatively, abnormal septal motion may be a consequence of loss of pericardial restraint, or even more simply translational error due to the anterior motion of the heart after pericardial incision (similar to those with a congenital absence of pericardium) which may resolve as the pericardium heals. Other thoughts are the motion may be due to the acute pericardial inflammation after surgery, with those that have prolonged abnormal septal motion (>1 year) suffering from a degree of constriction.[8]

Mitral stenosis

An exaggerated, early diastolic 'dip' (posterior motion) in the septum is noted in significant or severe mitral (see Figure 5 and Video S5), as well as reports of diastolic fluttering.[9] Severely diminished filling of the left ventricle creates a negative ventricular diastolic pressure reversing the transeptal pressure gradient. Furthermore, the high right-sided pressures that occur as a consequence of mitral stenosis also contribute to the change in transeptal pressure gradient. This pattern is likely only seen in severe mitral stenosis.

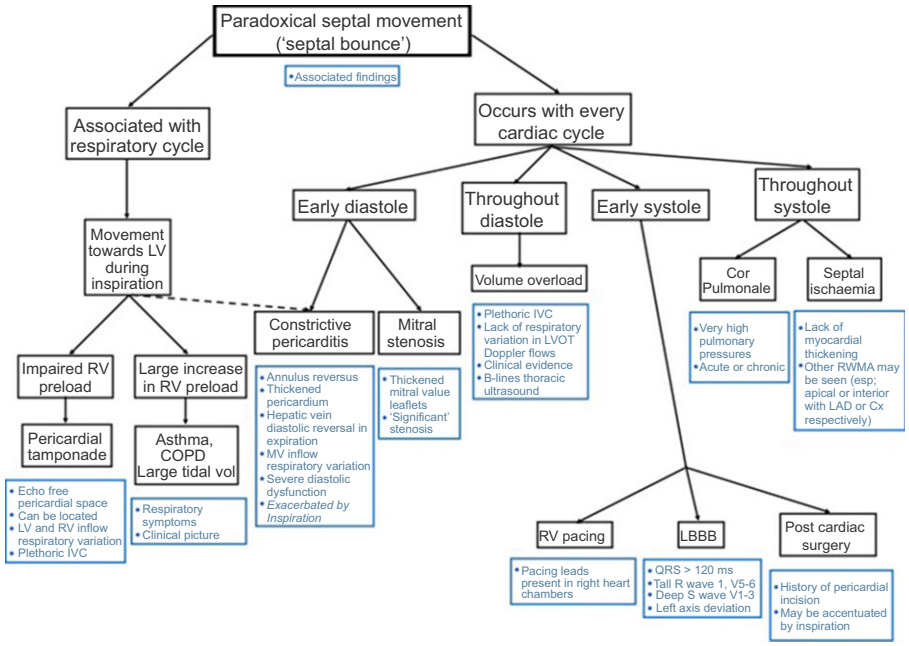

Figure 2: Systematic Approach to Paradoxical Septal Motion: Septal Bounce'. Aetiology and Associated Echocardiography and Clinical Findings.

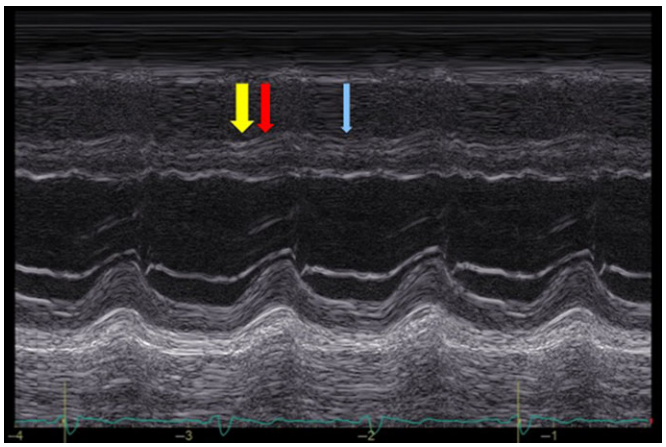

Figure 3: M-mode of Septal Motion During LBBB: Earlier Right Ventricle Contraction Before Left Ventricle Results in Early Systolic Posterior Septal Movement, Known Septal Beaking' (Yellow Arrow), then Flattening of the Septum as the Left Ventricle Contracts (red arrow) and Finally During Atrial Systole Septal Motion Posteriorly then Anteriorly due to the Tricuspid Valve Opening Before the Mitral (Blue Arrow).

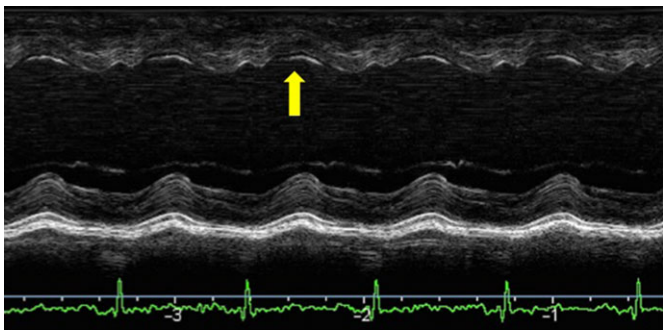

Figure 4: M-mode of Septal Motion after Open Cardiac Surgery. Abnormal Anterior Septal Motion with Systole (Yellow Arrows). (Image Courtesy of Associate Prof Stan Yastrebov).

Pericardial disease: constrictive pericarditis and tamponade

A key feature of septal movement in constrictive pericarditis and tamponade is the variation with respiration. In a normal physiological state, during inspiration, there is a reduction in intrathoracic pressure that results in minor RV and LV changes. The RV afterload is reduced and RV preload increased resulting in an increase in RV volume and blood flow. In contrast, LV preload is reduced; however, the negative intrathoracic pressure is transmitted through the pericardium and helps maintain the pressure gradient. In constrictive pericarditis and tamponade, there is reduced ventricular compliance due to a fixed pericardial volume and changes in intrathoracic pressure are not transmitted to the cardiac chambers. Hence, there is increased right ventricular filling on inspiration, with a significant reduction in left ventricular filling.[10] The reduction in left ventricle filling on inspiration is exaggerated by the pericardial restraint reducing overall ventricular compliance, and creating a fixed total cardiac volume. Hence, if right ventricular volume increases on inspiration, left ventricular volume has to fall, leading to posterior septal motion in early diastole (see Figure 6 and Video S6). The opposite is true in expiration. Hence, the classic ventricular interdependence whereby filling of one

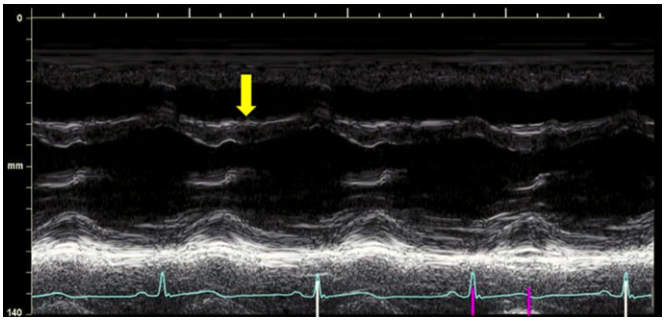

Figure 5: M-mode of Septum in Severe Mitral Stenosis: Impaired Left Ventricle Filling due to Mitral Stenosis Leads to Exaggerated Septal Diastolic Posterior Dip' (Yellow Arrows), Highlighting Unimpeded Right Ventricle Filling vs. the Left.

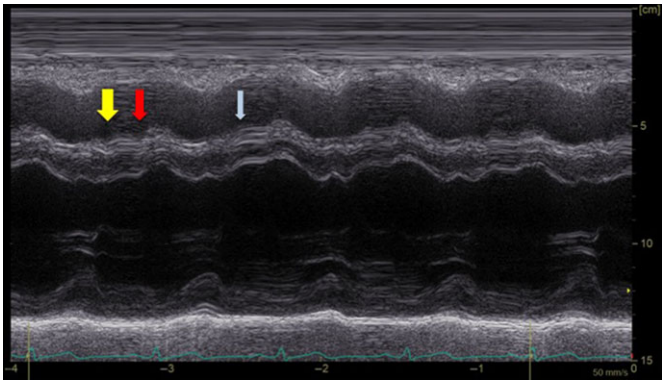

Figure 6: Abnormal Septal Motion in Constrictive Pericarditis: Diastolic (Posterior) Bowing of the Septum During Inspiration (Yellow Arrow), which is not Seen in the Subsequent Cardiac Cycle (Blue Arrow), with Subtle Double Wobble of the Septum (Red Arrow) Correlating with Atrial Contraction.

ventricle is dependent on the amount of blood flow into the other. There is evidence from simultaneous M-mode imaging and cardiac catheterisation that ventricular interdependence and relative transeptal pressure gradients create the abnormal septal motion.[11] The respiratory variation in abnormal septal movement (also referred to as septal shift or drift) in constrictive pericarditis is seen in 93% of cases.[12] Early diastolic filling is rapid in constrictive pericarditis, and ceases abruptly when the rigid pericardium cannot accommodate further fluid: hence, this abnormal early diastolic motion is transient. In tamponade, however, filling is impaired throughout diastole prolonging the abnormal early diastolic motion.

Another abnormal septal motion, also called 'diastolic shudder' or 'septal oscillations',[13] is evident in up to 96% of cases in mid to late diastole.[12] As the filling of the right ventricle abruptly ceases, the septum moves anteriorly after its initial bounce posteriorly, before again moving posteriorly: hence, the septum appears to oscillate or have a double wobble.[14] This may reflect the subtle interdependence created by timing differences in the opening of the mitral and tricuspid valves, and the final motion is attributable to atrial contraction into a non-compliant right ventricle. This motion is irrespective of respiration. In a series of constrictive pericarditis patients, septal excursion of greater than 12% with respiration detected on MRI had a 95% specificity for constrictive pericarditis, whereas multiple conditions have septal bounce.[15] Hence, one of the key features in constrictive pericarditis is the respiratory variation in the septum.

A recent study by Jogia *et al.*[14] correlated the severity of constriction with the above findings of septal movement on M-mode echocardiography. In mild and moderate constriction, only the single motion of the septum in early diastole is noted, that of the exaggerated septal shift with inspiration. In more severe cases, as the pericardium is more rigid, and hence filling of the RV is more rapid the 'double wobble' becomes also apparent.[16] In these cases, there was less exaggerated septal shift with inspiration. In most critical cases, there was complete diastolic posterior bowing of the septum (only one motion). Hence, whilst respiratory variation is obvious in mild to moderate constrictive pericarditis, it appears to be less of a feature in more severe cases.

Right ventricular pressure and volume overload

The RV is a highly compliant structure ejecting blood into a low-resistance pulmonary vasculature. Abrupt increases in pulmonary artery pressure or pulmonary vascular resistance, from any cause, results in increased RV afterload. The RV dilates easily under these conditions due to the highly compliant, thin RV free wall and as RV pressure increases, this leads to paradoxical (posterior) septal shift, predominantly during *systole* (see Figure 7a see Video S7a). In the extreme forms, this is known as cor pulmonale.

Pulmonary hypertension sees early systolic septal flattening as a consequence of the transient reversal of the transeptal pressure gradient (i.e. RV pressure is greater than LV before LV systole overcomes RV pressure). Furthermore, the septum is bowed due to changes in RV contraction as a result of increased RV afterload.[17] This leads to ongoing RV contraction after LV diastole begins[18,19] leading to early diastolic bowing due to the negative intraventricular pressure gradient across the septum.[20] A similar effect is also seen during mechanical ventilation in conditions such as ARDS where tidal volumes, PEEP or driving pressures[21] are too high leading to excessive RV afterload. The negative gradient and subsequent posterior septal bowing may be prolonged due to the subsequent atrial kick into the non-compliant RV, in what Mori *et al.* call 'Type A patterns',[22] and may also be associated with a reduced cardiac index. Furthermore, the concave nature of the septum at end diastole then gives the appearance of an anterior motion of the septum in early diastole. This type A configuration matches the initial descriptions of M-mode imaging of the septum in patients with pulmonary hypertension which described the motion to appear like the square root sign, which was shown to reflect the pressure gradient across the septum throughout the cardiac

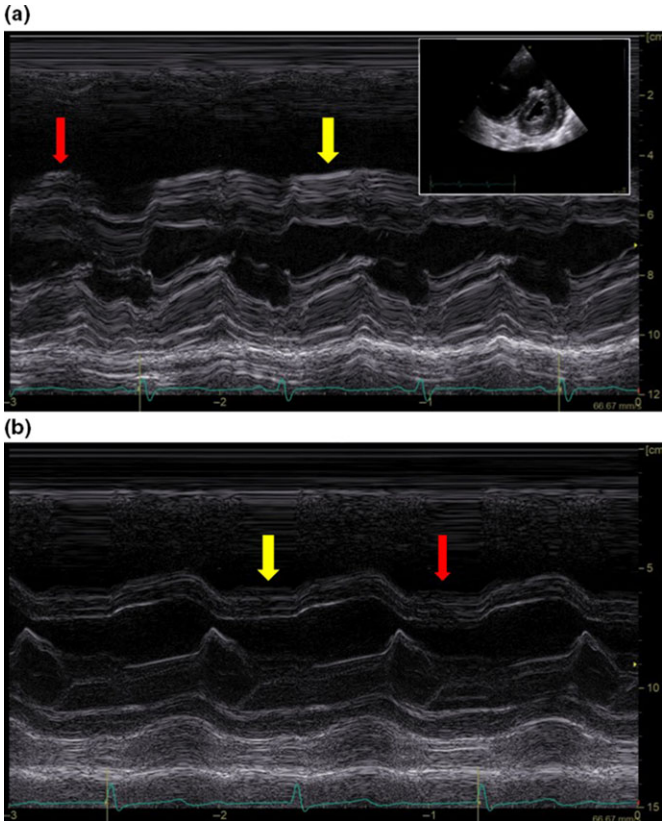

Figure 7: (a) Septal Motion in Right Ventricle Pressure Overload with Septal Flattening during Systole (Yellow Arrows) Worsened by Inspiration (Red Arrow); Inset B-mode Parasternal Long Axis Image at the Mid-papillary Level; (b) Volume Overload with Septal Flattening During Diastole (Yellow Arrows) Worsened by Inspiration (Red Arrow).

cycle.[20,23] 'Type B' patterns are described as having early diastolic bowing only and are considered more consistent with right ventricular volume overload (such as in a large atrial septal defect or severe tricuspid regurgitation) (see Figure 7b see Video S7b). Theoretically, pulmonary vasodilation may be able to change the septal motion from type A to type B.

Exaggerated inspiratory effort

Exaggerated inspiratory effort can significantly affect RV preload as well as afterload, and similarly the performance of the left ventricle. In conditions with abnormal increased negative intrapleural pressures, such as acute asthma or COPD, pleural pressures can be in excess of −35 mmHg.[24] This effect not only leads to large increases in RV preload, but also leads to increases in RV afterload through compression of pulmonary vasculature with alveolar hyperinflation. Furthermore, the exaggerated intrathoracic pressure increases LV transmural pressure and afterload.[25] Hence, whilst this may create a similar pattern to type B as described by Mori, it will vary with the respiratory cycle (increased RV volume and pressure on inspiration, reduced on expiration).

Haemodynamic consequences related to the presence of septal bounce

The septum is 25–40% of the ventricular muscle mass[26] and the normal mechanics of septal motion have been suggested to be vital for adequate function of both the left and also the right ventricle. Animal studies have demonstrated preservation of right ventricular function after inducing failure of the free wall, as long as the septum remains intact.[27] In regard to the left ventricle, histological and imaging studies in humans have suggested that LV twist and torsion contribute to up to 40% of systolic function; and the septum plays a key role.[28,29]

Buckberg argues that RV function is determined by the interaction of its lateral wall and the obliquely orientated fibres of the septum, which he nominates as the 'Lion of the Right ventricle',[26] citing MRI studies that indicate RV longitudinal function (upon which RV ejection and filling relies on) is due to the septal twisting and untwisting.[29] This may seem counterintuitive for echocardiography users who measure tricuspid annular plane systolic excursion (TAPSE) to monitor RV function under the premise that the majority of RV contraction is based on longitudinal motion of the lateral wall. Buckberg argues this longitudinal motion is in part due to septal shortening. In addition, he postulates that with increased pulmonary vascular resistance, the septum contributes more to the right ventricular contraction and as such the septum is the motor of biventricular function.[30]

Given the proposed shared function of the septum between the RV and LV creating a ventricular–ventricular interaction, a disease process of one ventricle will affect the other ventricle via the septum. This is also known as ventricular interdependence. Functional anatomical studies have been used to describe the effects on ventricular filling and ejection as a consequence of a change in septal myocardial fibre geometry. One example is the controversial helico-ventricular myocardial band model described by Torrent Guasp.[31] Septal dysfunction has been implicated when impaired LV filling is identified in the presence of severe pulmonary hypertension. In some studies, up to 88% of those with pulmonary hypertension and mean pulmonary artery pressure >50 mmHg had evidence of impaired LV relaxation.[32] Cardiac index has also been shown to reduce with escalating severity of pulmonary hypertension.[33] Potentially, this effect may be related to a change in fibre orientation in the septum leading to reducing LV filling and interference with LV contractility which lead to reduced cardiac output.[19]

Puwanant *et al.* showed that in pulmonary hypertension, the circumferential movement of the septum is impaired to a greater extent than that seen in the LV lateral wall, with a reduction in the overall torsion.[34] This septal dysfunction appeared to be due to changes in geometry rather than contractility: that is septal bowing and a non-circular LV configuration. They also demonstrated a close relationship between degree of septal displacement and degree of dysfunction. This highlights that the change in septal position may affect left ventricular filling and contraction independent of the haemodynamic

consequence of pulmonary hypertension. LV filling may also be impaired not only by ventricular interdependence, but also by the interventricular asynchrony created by prolonged right ventricular contraction.[18,35]

More readily apparent to the clinician are disruptions to the electrical timing causing disordered mechanical events, known as dyssynchrony. Bundle branch blocks and pacing alter the natural pattern of electromechanical coupling, but also the subsequent electrical conduction passing slowly although the ventricular myocardium rather than the usual specialised conduction pathway. Reduced pump function ensues as the coordinated and efficient contraction is altered: first disturbed intra-ventricular coupling (LV dyssynchrony) and secondly interventricular decoupling with resultant ventricular interdependence (characterised by septal bounce) due to the long delay in LV depolarisation relative to the RV. This is highlighted by the increased delay in closure between the aortic and pulmonary valves. In a series of patients with biventricular pacemakers that had underlying right and left bundle branch blocks, there was evidence of interventricular dyssynchrony in 75% of patients with LBBB compared with 8% of RBBB.[36]

The advent of biventricular pacing techniques has a particular role in highlighting septal dysfunction. In the previously described series of patients with biventricular pacing, there was a significant improvement in septal contraction, but none in the LV lateral wall, in patients with LBBB who underwent biventricular pacing. It should be noted there were subsequent improvements in diastolic dysfunction and stroke volume. Cardiac resynchronization allows the septum to move back to its central position, improving its functional orientation of fibres for contraction and also mitral leaflet coaptation.[37] The presence of septal flash in left bundle branch block indicates a higher likelihood that resynchronization therapy will benefit the patient with heart failure and long-standing atrial fibrillation.[38]

The consequences of septal dysfunction post-cardiothoracic surgery are controversial. Kang *et al.* describe a clinically insignificant change in LV function and the presence of abnormal septal motion being largely transient with only 15% of those identified having ongoing abnormal motion at one year[39] although these data are limited by the loss of follow-up to almost a third of patients and the limited use of some strain parameters. Alternatively, Roshanali *et al.*[40] found 97% of patients had ongoing abnormal septal motion at 12 months after coronary artery grafting, with an associated reduction in right ventricular longitudinal function (TAPSE, S' and RV free wall strain). Again, the helico-ventricular myocardial band model has been cited to suggest it is perhaps the right ventricle that suffers from changes to twisting and untwisting of the septum post-cardiothoracic surgery. Whilst markers of right ventricular function that rely on longitudinal motion have limited correlation with more global indices of right ventricular function[41] and have limited prognostic value[42] in the post-operative state, Nguyen *et al.*[43] argue that overall function will remain normal in spite of septal dysfunction as long as pulmonary vascular resistance is low. However, any further changes in right ventricular afterload will lead to right ventricular failure as the septum is unable compensate. Reduced global right ventricular function is associated with significantly reduced outcomes.[44] Right ventricular dysfunction post-left ventricular assist devices has also been linked to changes in septal geometry and function and is associated with a higher mortality[45] (although the change in septal function is likely a different mechanism to routine cardiothoracic surgery).

Our overall understanding of myocardial fibre architecture and cardiac mechanics is far from complete, and this includes the role of the septum in both LV and RV function. With advancing echocardiographic techniques (i.e. speckle tracking echocardiography) and other imaging modalities, our understanding of the function of the septum and subsequent changes in septal bounce should be further enhanced.

Conclusion

Septal bounce is a common abnormality seen in a variety of conditions, each of which may cause subtle differences in timing and severity. It can be challenging to analyse and a systematic approach is important. The pattern is best initially recognised on M-mode imaging, both to determine subtle movement as well as to discern accurate timing. Septal bounce has a potential effect on cardiac mechanics and may affect overall efficiency and performance. Further studies investigating the importance of the septum in cardiac function are warranted.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Video S1. Normal septal motion.

Video S2. Paradoxical septal motion from respiratory variation.

Video S3. Left bundle branch block.

Video S4. Post cardio-thoracic surgery septal bounce.

Video S5. Septal bounce from severe mitral stenosis.

Video S6. Septal bounce from constrictive pericarditis.

Video S7a. Paradoxical septal motion from pressure overloaded right ventricle.

Video S7b. Paradoxical septal motion from volume overloaded right ventricle.

